What is claimed is:

1. An antagonist that inhibits or an agonist that activates an activity a polypeptide selected from the group consisting of: a polypeptide comprising an amino acid sequence which is at least 90% identical to the amino acid sequence of SEQ ID NO:2, and a polypeptide comprising an amino acid sequence as set forth in SEQ ID NO:2, wherein said activity is selected from the group consisting of: NADPH-dependent reduction of acetoacetyl-acyl carrier protein (ACP) to generate β-hydroxyacyl-ACP; deprotonation of a group leading to a diminution in k_{cat}; deprotonation of a general acid responsible for donating a proton to the carbonyl oxygen during its reduction; binding of an ionizable group putatively binding the pyrophosphate bridge of NADPH; catalysis involving a lysine residue as a general acid; a conformational change inducing formation a low-barrier hydrogen bond (LBHB) in ketone reduction mechanism; a conformational change upon acetoacetyl-CoA binding resulting in formation of an LBHB; a conformational change upon acetoacetyl-CoA binding resulting in formation of an LBHB between Tyr157 and Lys161; energy provided from Tyr157 and Lys161; energy from forming an LBHB between Tyr157 and Lys161 facilitating proton transfer from Lys157 to the carbonyl oxygen; proton transfer from Lys157 to carbonyl oxygen; formation of an LBHB between Tyr157 and an Asp residue; strengthening of the role of Tyr157 in facilitating general acid catalysis; strengthening of the role of Tyr157 in facilitating general acid catalysis by Lys161; compression of active site; compression of active site resulting in the formation of a LBHB facilitating proton transfer to the carbonyl oxygen; hydride transfer from NADPH proceeding proton transfer from the Lys residue; formation of an anionic, tetrahedral reaction intermediate; formation of a charge-stabilized intermediate by protonated Lys group prior to proton transfer to form the β-hydroxy-keto product (**2**); and hydride transfer from NADPH proceeding proton transfer from the Lys residue, with an anionic, tetrahedral reaction intermediate (**1**) being formed, that is potentially charge-stabilized by protonated Lys group prior to proton transfer to form the β-hydroxy-keto product (**2**).

2. A method for the treatment of an individual having need to inhibit or activate Fab G polypeptide comprising the steps of: administering to the individual an antibacterially effective amount of an antagonist that inhibits or an agonist that activates an activity of a polypeptide selected from the group consisting of: a polypeptide comprising an amino acid sequence which is at least 90% identical to the amino acid sequence of SEQ ID NO:2, and a polypeptide comprising an amino acid sequence as set forth in SEQ ID NO:2, wherein said

activity is selected from the group consisting of: NADPH-dependent reduction of acetoacetyl-acyl carrier protein (ACP) to generate β-hydroxyacyl-ACP; deprotonation of a group leading to a diminution in k_{cat}; deprotonation of a general acid responsible for donating a proton to the carbonyl oxygen during its reduction; binding of an ionizable group putatively binding the pyrophosphate bridge of NADPH; catalysis involving a lysine residue as a general acid; a conformational change inducing formation a low-barrier hydrogen bond (LBHB) in ketone reduction mechanism; a conformational change upon acetoacetyl-CoA binding resulting in formation of an LBHB; a conformational change upon acetoacetyl-CoA binding resulting in formation of an LBHB between Tyr157 and Lys161; energy provided from Tyr157 and Lys161; energy from forming an LBHB between Tyr157 and Lys161 facilitating proton transfer from Lys157 to the carbonyl oxygen; proton transfer from Lys157 to carbonyl oxygen; formation of an LBHB between Tyr157 and an Asp residue; strengthening of the role of Tyr157 in facilitating general acid catalysis; strengthening of the role of Tyr157 in facilitating general acid catalysis by Lys161; compression of active site; compression of active site resulting in the formation of a LBHB facilitating proton transfer to the carbonyl oxygen; hydride transfer from NADPH proceeding proton transfer from the Lys residue; formation of an anionic, tetrahedral reaction intermediate; formation of a charge-stabilized intermediate by protonated Lys group prior to proton transfer to form the β-hydroxy-keto product (**2**); and hydride transfer from NADPH proceeding proton transfer from the Lys residue, with an anionic, tetrahedral reaction intermediate (**1**) being formed, that is potentially charge-stabilized by protonated Lys group prior to proton transfer to form the β-hydroxy-keto product (**2**).

3. A method for the treatment of an individual infected with a bacteria comprising the steps of administering to the individual an antibacterially effective amount of an antagonist that inhibits or an agonist that activates an activity of a polypeptide selected from the group consisting of: a polypeptide comprising an amino acid sequence which is at least 90% identical to the amino acid sequence of SEQ ID NO:2, and a polypeptide comprising an amino acid sequence as set forth in SEQ ID NO:2, wherein said activity is selected from the group consisting of: NADPH-dependent reduction of acetoacetyl-acyl carrier protein (ACP) to generate β-hydroxyacyl-ACP; deprotonation of a group leading to a diminution in k_{cat}; deprotonation of a general acid responsible for donating a proton to the carbonyl oxygen during its reduction; binding of an ionizable group putatively binding the pyrophosphate bridge of NADPH; catalysis involving a lysine residue as a general acid; a conformational change

inducing formation a low-barrier hydrogen bond (LBHB) in ketone reduction mechanism; a conformational change upon acetoacetyl-CoA binding resulting in formation of an LBHB; a conformational change upon acetoacetyl-CoA binding resulting in formation of an LBHB between Tyr157 and Lys161; energy provided from Tyr157 and Lys161; energy from forming an LBHB between Tyr157 and Lys161 facilitating proton transfer from Lys157 to the carbonyl oxygen; proton transfer from Lys157 to carbonyl oxygen; formation of an LBHB between Tyr157 and an Asp residue; strengthening of the role of Tyr157 in facilitating general acid catalysis; strengthening of the role of Tyr157 in facilitating general acid catalysis by Lys161; compression of active site; compression of active site resulting in the formation of a LBHB facilitating proton transfer to the carbonyl oxygen; hydride transfer from NADPH proceeding proton transfer from the Lys residue; formation of an anionic, tetrahedral reaction intermediate; formation of a charge-stabilized intermediate by protonated Lys group prior to proton transfer to form the β-hydroxy-keto product (**2**); and hydride transfer from NADPH proceeding proton transfer from the Lys residue, with an anionic, tetrahedral reaction intermediate (**1**) being formed, that is potentially charge-stabilized by protonated Lys group prior to proton transfer to form the β-hydroxy-keto product (**2**).

4. The method of claim 3 wherein said bacteria is selected from the group consisting of a member of the genus *Staphylococcus, Staphylococcus aureus,* a member of the genus *Streptococcus,* and *Streptococcus pneumoniae.*

5. A method for the treatment of an individual having need to inhibit or activate Fab G polypeptide comprising the steps of administering to the individual an antibacterially effective amount of an antagonist that inhibits or an agonist that activates an activity of Fab G selected from the group consisting of: NADPH-dependent reduction of acetoacetyl-acyl carrier protein (ACP) to generate β-hydroxyacyl-ACP; deprotonation of a group leading to a diminution in k_{cat}; deprotonation of a general acid responsible for donating a proton to the carbonyl oxygen during its reduction; binding of an ionizable group putatively binding the pyrophosphate bridge of NADPH; catalysis involving a lysine residue as a general acid; a conformational change inducing formation a low-barrier hydrogen bond (LBHB) in ketone reduction mechanism; a conformational change upon acetoacetyl-CoA binding resulting in formation of an LBHB; a conformational change upon acetoacetyl-CoA binding resulting in formation of an LBHB between Tyr157 and Lys161; energy provided from Tyr157 and Lys161; energy from forming an LBHB between Tyr157 and Lys161 facilitating proton transfer from

Lys157 to the carbonyl oxygen; proton transfer from Lys157 to carbonyl oxygen; formation of an LBHB between Tyr157 and an Asp residue; strengthening of the role of Tyr157 in facilitating general acid catalysis; strengthening of the role of Tyr157 in facilitating general acid catalysis by Lys161; compression of active site; compression of active site resulting in the formation of a LBHB facilitating proton transfer to the carbonyl oxygen; hydride transfer from NADPH proceeding proton transfer from the Lys residue; formation of an anionic, tetrahedral reaction intermediate; formation of a charge-stabilized intermediate by protonated Lys group prior to proton transfer to form the β-hydroxy-keto product (**2**); and hydride transfer from NADPH proceeding proton transfer from the Lys residue (Figure 5), with an anionic, tetrahedral reaction intermediate (**1**) being formed, that is potentially charge-stabilized by protonated Lys group prior to proton transfer to form the β-hydroxy-keto product (**2**).

6. A method for the treatment of an individual infected with a bacteria comprising the steps of administering to the individual an antibacterially effective amount of an antagonist that inhibits or an agonist that activates that activates an activity of Fab G selected from the group consisting of: NADPH-dependent reduction of acetoacetyl-acyl carrier protein (ACP) to generate β-hydroxyacyl-ACP; deprotonation of a group leading to a diminution in k_{cat}; deprotonation of a general acid responsible for donating a proton to the carbonyl oxygen during its reduction; binding of an ionizable group putatively binding the pyrophosphate bridge of NADPH; catalysis involving a lysine residue as a general acid; a conformational change inducing formation a low-barrier hydrogen bond (LBHB) in ketone reduction mechanism; a conformational change upon acetoacetyl-CoA binding resulting in formation of an LBHB; a conformational change upon acetoacetyl-CoA binding resulting in formation of an LBHB between Tyr157 and Lys161; energy provided from Tyr157 and Lys161; energy from forming an LBHB between Tyr157 and Lys161 facilitating proton transfer from Lys157 to the carbonyl oxygen; proton transfer from Lys157 to carbonyl oxygen; formation of an LBHB between Tyr157 and an Asp residue; strengthening of the role of Tyr157 in facilitating general acid catalysis; strengthening of the role of Tyr157 in facilitating general acid catalysis by Lys161; compression of active site; compression of active site resulting in the formation of a LBHB facilitating proton transfer to the carbonyl oxygen; hydride transfer from NADPH proceeding proton transfer from the Lys residue; formation of an anionic, tetrahedral reaction intermediate; formation of a charge-stabilized intermediate by protonated Lys group prior to proton transfer to form the β-hydroxy-keto product (**2**); and hydride transfer from NADPH proceeding proton

transfer from the Lys residue, with an anionic, tetrahedral reaction intermediate (**1**) being formed, that is potentially charge-stabilized by protonated Lys group prior to proton transfer to form the β-hydroxy-keto product (**2**).

7. The method of claim 6 wherein said bacteria is selected from the group consisting of: a member of the genus *Staphylococcus, Staphylococcus aureus,* a member of the genus *Streptococcus, and Streptococcus pneumoniae.*

8. A method for the treatment of an individual infected by *Streptococcus pneumoniae* comprising the steps of administering to the individual an antibacterially effective amount of an antagonist that inhibits or antagonist that activates an activity of *Streptococcus pneumoniae* Fab G selected from the group consisting of: NADPH-dependent reduction of acetoacetyl-acyl carrier protein (ACP) to generate β-hydroxyacyl-ACP; deprotonation of a group leading to a diminution in k_{cat}; deprotonation of a general acid responsible for donating a proton to the carbonyl oxygen during its reduction; binding of an ionizable group putatively binding the pyrophosphate bridge of NADPH; catalysis involving a lysine residue as a general acid; a conformational change inducing formation a low-barrier hydrogen bond (LBHB) in ketone reduction mechanism; a conformational change upon acetoacetyl-CoA binding resulting in formation of an LBHB; a conformational change upon acetoacetyl-CoA binding resulting in formation of an LBHB between Tyr157 and Lys161; energy provided from Tyr157 and Lys161; energy from forming an LBHB between Tyr157 and Lys161 facilitating proton transfer from Lys157 to the carbonyl oxygen; proton transfer from Lys157 to carbonyl oxygen; formation of an LBHB between Tyr157 and an Asp residue; strengthening of the role of Tyr157 in facilitating general acid catalysis; strengthening of the role of Tyr157 in facilitating general acid catalysis by Lys161; compression of active site; compression of active site resulting in the formation of a LBHB facilitating proton transfer to the carbonyl oxygen; hydride transfer from NADPH proceeding proton transfer from the Lys residue; formation of an anionic, tetrahedral reaction intermediate; formation of a charge-stabilized intermediate by protonated Lys group prior to proton transfer to form the β-hydroxy-keto product (**2**); and hydride transfer from NADPH proceeding proton transfer from the Lys residue, with an anionic, tetrahedral reaction intermediate (**1**) being formed, that is potentially charge-stabilized by protonated Lys group prior to proton transfer to form the β-hydroxy-keto product (**2**).

9. An antagonist that inhibits an activity of a polypeptide selected from the group consisting of: a polypeptide comprising an amino acid sequence which is at least 90% identical

to the amino acid sequence of SEQ ID NO:1, and a polypeptide comprising an amino acid sequence as set forth in SEQ ID NO:1, wherein said activity is selected from the group consisting of: NADPH-dependent reduction of acetoacetyl-acyl carrier protein (ACP) to generate β-hydroxyacyl-ACP; deprotonation of a group leading to a diminution in k_{cat}; deprotonation of a general acid responsible for donating a proton to the carbonyl oxygen during its reduction; binding of an ionizable group putatively binding the pyrophosphate bridge of NADPH; catalysis involving a lysine residue as a general acid; a conformational change inducing formation a low-barrier hydrogen bond (LBHB) in ketone reduction mechanism; a conformational change upon acetoacetyl-CoA binding resulting in formation of an LBHB; a conformational change upon acetoacetyl-CoA binding resulting in formation of an LBHB between Tyr157 and Lys161; energy provided from Tyr157 and Lys161; energy from forming an LBHB between Tyr157 and Lys161 facilitating proton transfer from Lys157 to the carbonyl oxygen; proton transfer from Lys157 to carbonyl oxygen; formation of an LBHB between Tyr157 and an Asp residue; strengthening of the role of Tyr157 in facilitating general acid catalysis; strengthening of the role of Tyr157 in facilitating general acid catalysis by Lys161; compression of active site; compression of active site resulting in the formation of a LBHB facilitating proton transfer to the carbonyl oxygen; hydride transfer from NADPH proceeding proton transfer from the Lys residue; formation of an anionic, tetrahedral reaction intermediate; formation of a charge-stabilized intermediate by protonated Lys group prior to proton transfer to form the β-hydroxy-keto product (**2**); and hydride transfer from NADPH proceeding proton transfer from the Lys residue, with an anionic, tetrahedral reaction intermediate (**1**) being formed, that is potentially charge-stabilized by protonated Lys group prior to proton transfer to form the β-hydroxy-keto product (**2**).

10. A method for the treatment of an individual having need to inhibit Fab G polypeptide comprising the steps of administering to the individual an antibacterially effective amount of an antagonist that inhibits an activity of a polypeptide selected from the group consisting of: a polypeptide comprising an amino acid sequence which is at least 90% identical to the amino acid sequence of SEQ ID NO:1, and a polypeptide comprising an amino acid sequence as set forth in SEQ ID NO:1, , wherein said activity is selected from the group consisting of: NADPH-dependent reduction of acetoacetyl-acyl carrier protein (ACP) to generate β-hydroxyacyl-ACP; deprotonation of a group leading to a diminution in k_{cat}; deprotonation of a general acid responsible for donating a proton to the carbonyl oxygen during

its reduction; binding of an ionizable group putatively binding the pyrophosphate bridge of NADPH; catalysis involving a lysine residue as a general acid; a conformational change inducing formation a low-barrier hydrogen bond (LBHB) in ketone reduction mechanism; a conformational change upon acetoacetyl-CoA binding resulting in formation of an LBHB; a conformational change upon acetoacetyl-CoA binding resulting in formation of an LBHB between Tyr157 and Lys161; energy provided from Tyr157 and Lys161; energy from forming an LBHB between Tyr157 and Lys161 facilitating proton transfer from Lys157 to the carbonyl oxygen; proton transfer from Lys157 to carbonyl oxygen; formation of an LBHB between Tyr157 and an Asp residue; strengthening of the role of Tyr157 in facilitating general acid catalysis; strengthening of the role of Tyr157 in facilitating general acid catalysis by Lys161; compression of active site; compression of active site resulting in the formation of a LBHB facilitating proton transfer to the carbonyl oxygen;
hydride transfer from NADPH proceeding proton transfer from the Lys residue; formation of an anionic, tetrahedral reaction intermediate; formation of a charge-stabilized intermediate by protonated Lys group prior to proton transfer to form the β-hydroxy-keto product (**2**); and hydride transfer from NADPH proceeding proton transfer from the Lys residue, with an anionic, tetrahedral reaction intermediate (**1**) being formed, that is potentially charge-stabilized by protonated Lys group prior to proton transfer to form the β-hydroxy-keto product (**2**).

11. A method for inhibiting an activity of Fab G polypeptide comprising the steps of contacting a composition comprising said polypeptide with an effective amount of an antagonist that inhibits an activity of Fab G, wherein said activity is selected from the group consisting of: NADPH-dependent reduction of acetoacetyl-acyl carrier protein (ACP) to generate β-hydroxyacyl-ACP; deprotonation of a group leading to a diminution in k_{cat}; deprotonation of a general acid responsible for donating a proton to the carbonyl oxygen during its reduction; binding of an ionizable group putatively binding the pyrophosphate bridge of NADPH; catalysis involving a lysine residue as a general acid; a conformational change inducing formation a low-barrier hydrogen bond (LBHB) in ketone reduction mechanism; a conformational change upon acetoacetyl-CoA binding resulting in formation of an LBHB; a conformational change upon acetoacetyl-CoA binding resulting in formation of an LBHB between Tyr157 and Lys161; energy provided from Tyr157 and Lys161; energy from forming an LBHB between Tyr157 and Lys161 facilitating proton transfer from Lys157 to the carbonyl oxygen; proton transfer from Lys157 to carbonyl oxygen; formation of an LBHB between

Tyr157 and an Asp residue; strengthening of the role of Tyr157 in facilitating general acid catalysis; strengthening of the role of Tyr157 in facilitating general acid catalysis by Lys161; compression of active site; compression of active site resulting in the formation of a LBHB facilitating proton transfer to the carbonyl oxygen; hydride transfer from NADPH proceeding proton transfer from the Lys residue; formation of an anionic, tetrahedral reaction intermediate; formation of a charge-stabilized intermediate by protonated Lys group prior to proton transfer to form the β-hydroxy-keto product (**2**); and hydride transfer from NADPH proceeding proton transfer from the Lys residue, with an anionic, tetrahedral reaction intermediate (**1**) being formed, that is potentially charge-stabilized by protonated Lys group prior to proton transfer to form the β-hydroxy-keto product (**2**).

12. A method for inhibiting an activity of Fab G, wherein said activity is selected from the group consisting of: NADPH-dependent reduction of acetoacetyl-acyl carrier protein (ACP) to generate β-hydroxyacyl-ACP; deprotonation of a group leading to a diminution in k_{cat} (Figure 1A); deprotonation of a general acid responsible for donating a proton to the carbonyl oxygen during its reduction; binding of an ionizable group putatively binding the pyrophosphate bridge of NADPH; catalysis involving a lysine residue as a general acid; a conformational change inducing formation a low-barrier hydrogen bond (LBHB) in ketone reduction mechanism; a conformational change upon acetoacetyl-CoA binding resulting in formation of an LBHB; a conformational change upon acetoacetyl-CoA binding resulting in formation of an LBHB between Tyr157 and Lys161; energy provided from Tyr157 and Lys161; energy from forming an LBHB between Tyr157 and Lys161 facilitating proton transfer fom Lys157 to the carbonyl oxygen; proton transfer from Lys157 to carbonyl oxygen; formation of an LBHB between Tyr157 and an Asp residue; strengthening of the role of Tyr157 in facilitating general acid catalysis; strengthening of the role of Tyr157 in facilitating general acid catalysis by Lys161; compression of active site; compression of active site resulting in the formation of a LBHB facilitating proton transfer to the carbonyl oxygen; hydride transfer from NADPH proceeding proton transfer from the Lys residue; formation of an anionic, tetrahedral reaction intermediate; formation of a charge-stabilized intermediate by protonated Lys group prior to proton transfer to form the β-hydroxy-keto product (**2**); and hydride transfer from NADPH proceeding proton transfer from the Lys residue, with an anionic, tetrahedral reaction intermediate (**1**) being formed, that is potentially charge-stabilized by protonated Lys group prior to proton transfer to form the β-hydroxy-keto product (**2**).

13. The method of claim 12 wherein said bacteria is selected from the group consisting of: a member of the genus *Staphylococcus, Staphylococcus aureus,* a member of the genus *Streptococcus,* and *Streptococcus pneumoniae.*

14. A method for inhibiting a growth of bacteria comprising the steps of contacting a composition comprising bacteria with an antibacterially effective amount of an antagonist that inhibits an activity of Fab G, wherein said activity is selected from the group consisting of: NADPH-dependent reduction of acetoacetyl-acyl carrier protein (ACP) to generate β-hydroxyacyl-ACP; deprotonation of a group leading to a diminution in k_{cat}; deprotonation of a general acid responsible for donating a proton to the carbonyl oxygen during its reduction; binding of an ionizable group putatively binding the pyrophosphate bridge of NADPH; catalysis involving a lysine residue as a general acid; a conformational change inducing formation a low-barrier hydrogen bond (LBHB) in ketone reduction mechanism; a conformational change upon acetoacetyl-CoA binding resulting in formation of an LBHB; a conformational change upon acetoacetyl-CoA binding resulting in formation of an LBHB between Tyr157 and Lys161; energy provided from Tyr157 and Lys161; energy from forming an LBHB between Tyr157 and Lys161 facilitating proton transfer from Lys157 to the carbonyl oxygen; proton transfer from Lys157 to carbonyl oxygen; formation of an LBHB between Tyr157 and an Asp residue; strengthening of the role of Tyr157 in facilitating general acid catalysis; strengthening of the role of Tyr157 in facilitating general acid catalysis by Lys161; compression of active site; compression of active site resulting in the formation of a LBHB facilitating proton transfer to the carbonyl oxygen; hydride transfer from NADPH proceeding proton transfer from the Lys residue; formation of an anionic, tetrahedral reaction intermediate; formation of a charge-stabilized intermediate by protonated Lys group prior to proton transfer to form the β-hydroxy-keto product (**2**); and hydride transfer from NADPH proceeding proton transfer from the Lys residue, with an anionic, tetrahedral reaction intermediate (**1**) being formed, that is potentially charge-stabilized by protonated Lys group prior to proton transfer to form the β-hydroxy-keto product (**2**).

15. The method of claim 14 wherein said bacteria is selected from the group consisting of: a member of the genus *Staphylococcus, Staphylococcus aureus,* a member of the genus *Streptococcus,* and *Streptococcus pneumoniae.*

16. A method for inhibiting a Fab G polypeptide comprising the steps of contacting a composition comprising bacteria with an antibacterially effective amount of an antagonist that inhibits an activity of Fab G, wherein said activity is selected from the group consisting of:

NADPH-dependent reduction of acetoacetyl-acyl carrier protein (ACP) to generate β-hydroxyacyl-ACP; deprotonation of a group leading to a diminution in k_{cat}; deprotonation of a general acid responsible for donating a proton to the carbonyl oxygen during its reduction; binding of an ionizable group putatively binding the pyrophosphate bridge of NADPH; catalysis involving a lysine residue as a general acid; a conformational change inducing formation a low-barrier hydrogen bond (LBHB) in ketone reduction mechanism; a conformational change upon acetoacetyl-CoA binding resulting in formation of an LBHB; a conformational change upon acetoacetyl-CoA binding resulting in formation of an LBHB between Tyr157 and Lys161; energy provided from Tyr157 and Lys161; energy from forming an LBHB between Tyr157 and Lys161 facilitating proton transfer from Lys157 to the carbonyl oxygen; proton transfer from Lys157 to carbonyl oxygen; formation of an LBHB between Tyr157 and an Asp residue; strengthening of the role of Tyr157 in facilitating general acid catalysis; strengthening of the role of Tyr157 in facilitating general acid catalysis by Lys161; compression of active site; compression of active site resulting in the formation of a LBHB facilitating proton transfer to the carbonyl oxygen; hydride transfer from NADPH proceeding proton transfer from the Lys residue; formation of an anionic, tetrahedral reaction intermediate; formation of a charge-stabilized intermediate by protonated Lys group prior to proton transfer to form the β-hydroxy-keto product (**2**); and hydride transfer from NADPH proceeding proton transfer from the Lys residue, with an anionic, tetrahedral reaction intermediate (**1**) being formed, that is potentially charge-stabilized by protonated Lys group prior to proton transfer to form the β-hydroxy-keto product (**2**).

17. The method of claim 16 wherein said bacteria is selected from the group consisting of: a member of the genus *Staphylococcus, Staphylococcus aureus,* a member of the genus *Streptococcus,* and *Streptococcus pneumoniae.*